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Case Report Open Access

Case Report of Transient Global Amnesia

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A 62 y/o female with no significant past medical history, presented with confusion, disorientation and short- term memory loss for approximately one hour prior to arrival in the Emergency Department (ED). She recalled driving to work and then suddenly felt like she was in a "dream-like state", and could not recall any events since that time. She arrived at work, where a security guard found her wandering around confused and 911 was called. EMS arrived to find patient on the bathroom floor, disoriented. On arrival to the ED, she was alert but oriented only to self. She kept repeating the same questions: "Am I having a dream?", and "How did I get here?" She was feeling well and acting appropriate the night before, according to her husband. She could, with some effort and concentration, remember the names of her husband and children. She took no medications, and family history was negative for stroke or seizure.

Complete 14-point review of systems was negative including fever, headache, visual changes, and loss of consciousness, automatisms, night sweats, rashes, and insect bites. Vital signs were normal. Physical exam including extensive neurological examination with fundoscopy showed no abnormalities. On mental status exam, the patient was alert but oriented to name only, recall was 1/3 at 5 minutes, and Mental Status Exam was otherwise unremarkable.

Extensive laboratory data was normal with the exception of a slightly low Folate (5.5) and Vitamin B12 (394).

Imaging: CT head was performed within an hour of arrival and negative for acute pathology. An MRI was performed 22 hours after initial presentation, which showed a 4mm abnormal focus of restricted diffusion along lateral posterior aspect of the left hippocampus, representing an acute abnormality of the hippocampus. There were no chronic white matter ischemic changes. EEG showed a nonspecific temporal lobe abnormality with no epileptiform activity.

The differential diagnosis for Transient Global Amnesia (TGA) includes complex partial seizures, a post-ictal state, psychogenic amnesia, cerebro vascular insufficiency or thrombus, migraine, toxin or drug effect, head injury, metabolic abnormality, high altitudes, tumor, cerebral bleed, or encephalitis (Figure 1) [1-3].

TGA is defined as the sudden-onset of anterograde memory loss, with impairment of orientation in space and time and inability to form new memories or retain new information, usually in persons >50. There are no associated neurological deficits. It can last anywhere from 1 to 24 hours, but complete symptom resolution generally occurs between 9-11 hours [3-5]. It is common for patients to be oriented to self but not to time or place, as with our patient. Consciousness remains undisturbed, and patients are still able to carry out complex tasks. Motor skills, speech, language, personality, judgment and memory registration also remain intact. Due to the sudden-onset of symptoms, anxiety is common, and it is typical for patients to perseverate, asking the same questions repeatedly- often with similar gestures and vocal intonation [1,3,6]. Incidence has been estimated to be 5 to 32 per 100,000 people annually [1,6].

Through case reports, a number of precipitating factors have been identified, which include: vigorous exercise, sudden temperature change, and emotional instability- although none of these were present in our patient [6].

CT head is the preferred initial study when a patient presents with an acute focal neurological deficit, and is typically negative in TGA. MRI brain is essential to rule-out other causes of amnesia, and will detect foci of restricted diffusion in the hippocampus, common in TGA. EEG can also help rule-out an alternate diagnosis. In TGA, the most common, but rarely seen finding on EEG is temporal lobe dysfunction.

Several etiologies of TGA have been implicated, including arterial thrombo embolism or paradoxical embolism, vertebral angiography or other medical procedures, physical exercise, sexual intercourse, emotional stress, extremes in temperature, neck hyperextension, cerebral ischemic disease, migraine, spreading cortical depression, psychological disturbances, venous ischemia, and jugular valve incompetence [1,2]. Studies so far have revealed no clear consensus on either etiology or pathogenesis of TGA.

There is consensus, however, that the parts of the brain affected are likely to involve the mediobasal temporal region, hippocampus, and parahippo campus- all of which play a central role in memory formation [6]. In this patient's MRI, we see a 4 mm focus of restricted diffusion along the lateral posterior aspect of the left hippocampus. Mesiotemporal and hippocampal punctate lesions on MRI with DWI have been described, and are present in 11-84% of TGA cases [2,7,8]. A single DWI lesion is categorized as a nonspecific finding with several

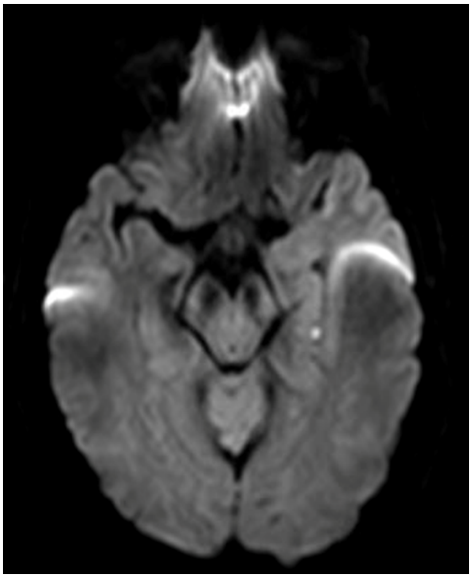

Figure 1: Transient Global Amnesia MRI.

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possible underlying mechanisms, all leading to local energy failure. It is theorized that these lesions may not necessarily be infarctions but are instead infarct-like abnormalities, and not necessarily due to vascular disease [2]. Studies comparing vascular risk profiles in TGA patients vs. normal age and sex-matched patients show no statistically significant difference, suggesting something other than a hypoxic-ischemic etiology of TGA [2]. Still, others contend that these MRI lesions support an ischemic etiology [7].

The CA1 region of the hippocampus has been described as and this area commonly shows lesions in TGA [7,8]. Some postulate that because the hippocampus is profoundly sensitive to pressure changes, venous ischemia can easily occur in this area without other neurologic symptoms [6].

After six hours this patient was back at neurologic baseline and all symptoms had resolved, while her amnesia of the event persisted. The patient had a follow- up appointment with a neurologist 1 month later, and did not report recurrence of symptoms, or any new neurologic symptoms. She denied any episodes of losing track of time, confusion or disorientation.

TGA is a remarkable, but rare and benign condition. It is frequently missed because the diagnosis is rarely considered and the clinical picture is poorly recognized. No formal treatment or follow-up is required. In similar case presentations, it is critical to obtain an accurate and detailed history from a reliable observer, perform a thorough neurologic exam, and rule out a more pathological cause of amnesia, specifically cerebro vascular accidents.

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